

**In the United States Court of Federal Claims  
OFFICE OF SPECIAL MASTERS  
No. 20-672V**

*Scott Rooney, Nemes Rooney, P.C., Farmington Hills, MI, for Petitioner.*

Lynn Schlie, U.S. Dep't of Justice, Washington, DC, for Respondent.

## **ENTITLEMENT DECISION<sup>1</sup>**

On June 2, 2020, Richard Jaye filed a petition for compensation under the National Vaccine and Injury Compensation Program (the “Vaccine Program”)<sup>2</sup> (ECF No. 1), alleging that a pneumococcal vaccine he received on December 8, 2017, caused him to incur Guillain-Barré syndrome (“GBS”). (Petitioner previously alleged that a flu vaccine administered to him in early October of that same year may also have been causal, but seems to have abandoned that aspect of his claim).

Because of my prior experience resolving Vaccine Program claims asserting a similar causation theory, I determined that the matter could reasonably be resolved via ruling on the record, and to that end the parties have filed briefs in support of their respective positions. See Petitioner’s Motion, dated September 29, 2023 (ECF No. 81-1) (“Mot.”); Respondent’s

<sup>1</sup> Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction “of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy.” Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id.*

<sup>2</sup> The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter “Vaccine Act” or “the Act”]. Individual section references hereafter will be to Section 300aa of the Act (but will omit the statutory prefix).

Opposition, dated January 30, 2024 (ECF No. 86) (“Opp.”); Petitioner’s Reply, dated March 6, 2024 (ECF No. 88) (“Reply”).

Having reviewed the above plus the filed medical records, expert reports, and associated literature (including items filed only in connection with this claim), I hereby deny an entitlement award. As discussed in greater detail below, Petitioner has not preponderantly established that the pneumococcal vaccine “can cause” GBS—and this alone is grounds for dismissal. I have reached the same determination in several prior Program cases, based on a comparable theory, and no scientific or medical evidence offered *in this case* that I have not previously considered supports an alternative finding, or reflects new scientific/medical developments suggesting that rejection of the theory should be revisited.

## I. Factual Background

The relevant facts can be briefly summarized. Petitioner (then 72 years old) received the pneumococcal vaccine on December 5, 2017, in the context of an annual physical exam. Ex. 17 at 33. Less than two weeks later, he sought emergency care due to two days of chest pain (coupled with bilateral leg pain). Ex. 4 at 27. Then, on December 19, 2017 (now two weeks post-vaccination), he informed his primary care physician that he was experiencing generalized arthralgias and myalgias, plus difficulty walking. Ex. 17 at 34. A few days thereafter, he sought treatment from a physical medicine and rehabilitation specialist for arm and leg pain, weakness, and difficulty standing and maintaining balance. Ex. 12 at 23–24. He also saw a neurologist on December 22, 2017, at which time he specifically reported onset of weakness on December 13<sup>th</sup>. Ex. 3 at 11–12.

Based on exam and presentation, the neurologist opined Petitioner might be experiencing GBS. Ex. 3 at 11–12. Petitioner subsequently went to the emergency room, where cerebrospinal fluid testing produced results consistent with GBS. Ex. 4 at 104–09. He was then hospitalized, receiving immunodeficiency treatments and later in-patient rehab. Petitioner’s diagnosis was refined over time to be an axonal form of GBS, and he continued to experience sequelae from the disease into 2019 and even later. Ex. 14-1 at 83; Ex. 13 at 16–19, 79–80; Ex. 12 at 35–36.

## II. Expert Reports

### A. Petitioner’s Expert – Lawrence Steinman, M.D.

Dr. Steinman, a neurologist, prepared two written reports for the Petitioner. Report, dated July 26, 2022, filed as Ex. 28 (ECF No. 68-1) (“First Steinman Rep.”); Report, dated April 2, 2023, filed as Ex. 29 (ECF No. 78-1) (“Second Steinman Rep.”).

As shown in his CV, Dr. Steinman received his undergraduate degree from Dartmouth College, and his medical degree from Harvard Medical School. *Curriculum Vitae*, filed as Ex. 28

Ref. 3 (ECF No. 70-3) (“Steinman CV”) at 1. He then completed residencies in neurology and pediatrics at Stanford University. *Id.* He has worked as a professor of neurology and pediatrics at Stanford for the past 40 years. *Id.*; Steinman First Rep. at 1. He is board certified in neurology from the American Board of Psychiatry and Neurology. Steinman CV at 2. Dr. Steinman has also published hundreds of peer-reviewed publications on neurology and autoimmune diseases. *Id.* at 6–49. He holds several patents related to the diagnosis and treatment of autoimmune and demyelinating diseases. *Id.* at 2–3. He presently serves as the George A. Zimmerman Professor of Neurological Sciences, Neurology, Genetics and Pediatrics at Stanford University. *Id.* at 1.

The opinions expressed in Dr. Steinman’s two reports (especially his initial report) are consistent in all material ways to what was offered—*also by Dr. Steinman*—in two prior cases I decided, also involving the contention that the pneumococcal vaccine can cause GBS. See *Gamboa-Avila v. Sec'y of Health & Hum. Servs.*, No. 18-925V, 2023 WL 6536207, at \*2-10 (Fed. Cl. Spec. Mstr. Sept. 11, 2023), *mot. for review den'd*, 170 Fed. Cl. 441 (2024), *appeal docketed*, No. 24-1765 (Fed. Cir. May 1, 2024); *Trollinger v. Sec'y of Health & Hum. Servs.*, No. 16-473V, 2023 WL 2521912, at \*25 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for review den'd*, No. 16-VV-473, 2023 WL 5249583 (Fed. Cl. 2023).

The similarity of Dr. Steinman’s opinions offered in this case with the opinions he offered in *Gamboa-Avila* and *Trollinger* is unmistakable—in ways large and small. For example, in reports filed in each of these cases, Dr. Steinman included a graphic from his seminal *Scientific American* article about molecular mimicry (a graphic that he likely has included in *every one* of the hundreds of reports filed in the Program).<sup>3</sup> More specific to the causation theory at issue, the reports filed herein also include the previously-offered contentions that (a) phospholipids are found in the antigenic components of the pneumococcal vaccine, (b) GBS patients can be shown to possess antibodies to these antigens, (c) “polar head groups” on the myelin sheath covering nerves are putative targets for the antibodies, and (d) the vaccine’s conjugate component (which is included in the formulation almost wholly to increase its immunogenicity, rather than to engender immunity *per se* to it) contains a mimic to Contactin-1, a protein also found in myelin.<sup>4</sup>

In addition, all three report “collections”<sup>5</sup> contain virtually identical explanations for Dr. Steinman’s “filtration” methodology for finding the degree of observed amino acid homology evidentiarily significant, in defense of Dr. Whitton’s contentions that his showing of molecular mimicry is not enough to render his theory preponderantly convincing. First Steinman Rep at 25–

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<sup>3</sup> Compare First Steinman Report at 9 to Dr. Steinman’s first report in *Gamboa-Avila*. (ECF No. 38-1). (I recognize that the *Gamboa-Avila* filings are not evidence in this case, but cite to them solely to substantiate that the opinion Dr. Steinman has previously offered is consistent with what he proposes herein).

<sup>4</sup> Compare First Steinman Rep. at 9–19 with *Gamboa-Avila* ECF No. 38-1 at 10–20.

<sup>5</sup> In this case, two reports authored by Dr. Steinman were filed – whereas in *Gamboa-Avila* and *Trollinger*, four reports were prepared and filed. *Gamboa-Avila*, 2023 WL 6536207, at \*2; *Trollinger*, 2023 WL 2521912, at \*3.

29. And an informal comparison of literature cited in the reports from all three cases reveals that *more than 20 items* identical to those he also offered in these two prior cases have been cited in this case as well.<sup>6</sup>

Because of the foregoing, I do not include a detailed summary of the opinions contained in Dr. Steinman’s reports filed in this matter—as I have already repeatedly considered *the same exact theory*. See, for example, *Gamboa-Avila*, 2023 WL 6536207, at \*26–29; *Trollinger*, 2023 WL 2521912, at \*27–30. As before, Dr. Steinman maintains that an autoimmune cross-reaction stimulated by components of the pneumococcal vaccine was sufficient to cause the demyelination characteristic of GBS. He specifically argues that the pneumococcal vaccine contains a “molecular mimic” relevant to the pathogenesis of GBS, the “polar head group of the phosphoglycerol and phosphocholine molecules.” First Steinman Rep. at 19. Because of sequential/structural identity in a chain of molecules making up that group and a chain found in components of the vaccine, phospholipid structures in the nerve myelin are mistakenly attacked by the vaccine-generated antibodies, leading to pathology. *Id.* And these antibodies, he maintains, are often found in the context of GBS (although Dr. Steinman struggles to show they are primarily, initially, or in any manner *at all* pathogenic of GBS, as opposed to being merely a measurable by-product of a disease process that has a different primary cause). Dr. Steinman also argued that the vaccine’s CRM<sub>197</sub> conjugate contains mimicking molecules for a different myelin protein, Contactin, and thus act as a separate basis for the proposed cross-reaction (even though that conjugate is not included in the vaccine with the goal of stimulating the production of antibodies, but instead merely improves the vaccine’s immunogenicity). *Id.*

Admittedly, (and perhaps partially owing to the fact that his second report was prepared in the spring of 2023—and hence reflects a slight evolution of his thinking on the theory presented in this case, in comparison to earlier matters), Dr. Steinman has offered some “newer” (meaning not previously referenced, as opposed to recently published) items of medical or scientific literature in support of his arguments, refining a few of his arguments in the process. But none appreciably aid the core theory offered.

For example, Dr. Steinman has cited some items of literature I have not previously confronted to show that (contrary to Dr. Whitton’s contention that molecular mimics abound in nature, but do not commonly result in autoimmune disease) in some cases experiments where direct mimics were introduced into humans *resulted* in disease. See Second Steinman Rep. at 3; L. Kappos et al., *Induction of Non-Encephalitogenic Type 2 T Helper-Cell Autoimmune Response in Multiple Sclerosis After Administration of Altered Peptide Ligand in a Placebo-Controlled, Randomized Phase II Trial*, 6 Nat. Med. 1176 (2000), filed as Ex. 29-1 (ECF No. 90-1) (“Kappos”); B. Bielekova et al., *Encephalitogenic Potential of the Myelin Basic Protein Peptide*

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<sup>6</sup> Compare First Steinman Rep. at 32–34, with *Gamboa-Avila*, ECF No. 38-1 at 22–23.

(*Amino Acids 83-99) in Multiple Sclerosis: Results of a Phase II Trial with an Altered Peptide Ligand*, 6 Nat. Med. 1167 (Oct. 2020), filed as Ex. 29-2 (ECF No. 90-2) (“Bielekova”).

But these items of literature are distinguishable, or only indirectly support the causal theory Dr. Steinman champions. Kappos, for example, was focused on Multiple Sclerosis (“MS”—a central nervous system demyelinating disease that is largely distinguishable from GBS, even though both involve demyelination<sup>7</sup>—and specifically experimental efforts to *positively* regulate the response to an autoimmune attack at the likely site of demyelination, which in turn involved how certain immune cells react to myelin basic protein, a nerve myelin “building block.” Kappos at 1176, 1177. Its authors directly immunized 142 patients who already were diagnosed with MS with either placebos or varying amounts of an altered peptide ligand (“APL”) that was theorized to induce an ameliorative cross-reaction that would reduce the likelihood of clinical relapses. *Id.* at 1177. Because of concerns about observed adverse reactions to the experimental treatment, the study was halted, but Kappos’s authors concluded based on data they initially collected that the induced autoimmune response (which largely involved T cells—not B cell-production of antibodies) might be a helpful treatment for MS. *Id.* at 1180. Thus, Kappos actually stands for the proposition that mimicry might (more often than not) result in *beneficial autoimmunity*, rather than the generalized and unsubstantiated dangers Dr. Steinman seeks to establish. And since it involved individuals who were already suffering from MS, the impact of receipt of the APL directly is not the same as the contention that a single vaccine might *instigate* disease through unplanned cross-reactions attributable to molecular mimicry.

Bielekova was published in the same journal (even issue) as Kappos, and also deals with experimental ways of introducing altered peptide ligands designed to cross-react, positively, with MBP demyelination situses in the treatment of MS. Bielekova at 1167. It involved a smaller pool of subjects than Kappos (24 patients), and it too resulted in discontinuation due to poor tolerance of the treatment. *Id.* at 1167. This study’s results also either observed no improvements in disease course, or (in a small subset of patients) worsening. *Id.* at 1172. Ultimately, Bielekova’s authors concluded that while “target antigens” identified in other kinds of studies could have experimental significance when applied to *in vivo* study efforts, “a better understanding of the complexities of the pathogenesis of T-cell mediated autoimmune disease” was needed in attempting to design ameliorative immunotherapies. *Id.* at 1173. As with Kappos, Dr. Steinman’s reference to Bielekova seems aimed at underscoring that experimental and directed “immunization” can produce cross-reactions specific to nerve myelin. But there is a wide gulf between the kind of experimentally-targeted immunizations at issue in these studies, and the receipt of a vaccine

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<sup>7</sup> MS is a chronic condition, not acute and monophasic like GBS; it impacts the spine and brain, not peripheral nerves; and to date only the Epstein-Barr Virus has been associated with its development, whereas other kinds of viral and bacterial infections are associated with GBS. More significantly for present purposes, MS is generally thought to be mediated by T cell attacks—not cross-reacting auto-antibodies produced by B cells, which any vaccine causation theory would rely upon (since antibody production is an initial, prime goal of vaccination). Kappos at 1176.

(which is not designed at all to react in this way)—and it is glib to elide vaccination and immunization in the way Dr. Steinman does.

Dr. Steinman also offers an older item of literature to rebut Dr. Whitton’s contention that the size of the phosphoglycerol molecule is likely too small to prompt cross-reaction. Second Steinman Rep. at 4; E. Barbar et al., *Binding of Phenylphosphocholine-Carrier Conjugates to the Combining Site of Antibodies Maintains a Conformation of the Hapten*, 35 Biochem. 2958 (1996), filed as Ex. 29-3 (ECF No. 90-3) (“Barbar”). Barbar (authored nearly 30 years ago, but without demonstrated follow-up in the literature confirming its importance or even its findings) sought in general to evaluate the role of the protein “carrier” that many vaccines feature, and what impact the carrier will have on antibody production in response to the smaller, “hapten”<sup>8</sup> molecules attached to the protein. Barbar at 2966. The pneumococcal vaccine at issue in this case includes a protein carrier/conjugate, which is intended to assist the immune process even though the bacterial polysaccharides conjugated to it are intended to be the primary antigenic components. *Package Insert: Prevnar-13* at 25, filed on October 6, 2022 as Ex. 28-10 (ECF No. 70-11).

But Barbar was not a study of the pneumococcal vaccine—or even *any* vaccine designed to protect against *S. Pneumoniae*. Indeed, Barbar’s authors noted that “the choice of haptens was made in order to evaluate the contribution of the carrier to binding, and its effect on hapten conformation in the active site [of that same binding].” Barbar at 2958. Thus, although Dr. Steinman is accurate in citing Barbar for the limited fact that it involves a phosphocholine molecule, the study does not make it more or less likely that comparable components of the pneumococcal vaccine produce a cross-reaction to demyelinating target sites on the nerve myelin. In fact, were it the case that an article published nearly 30 years ago stood for the proposition that antibody attacks on, or cross-reactions in association with, phosphoglycerol were possible despite the molecule’s size, then it is a mystery why the association Dr. Steinman gleans (which suggests an alternative cause for GBS) is not recognized more widely. Barbar thus offers limited additional support for Petitioner’s causation theory.

Another set of previously-unfiled items of literature are invoked by Dr. Steinman as “showing that phospholipids are targeted in GBS, including very early in the disease.” Second Steinman Rep. at 6, 7-11; T. Al-Temeemi et al., *Antiphospholipid Antibody in Serum of Guillain-Barré Syndrome Patients*, 10 Iraqi J. Med. Sci. 191 (2012), filed on May 9, 2024 as Ex. 29-4 (ECF No. 90-4) (“Al-Temeemi”); J. Terryberry et al., *Myelin- and Microbe-Specific Antibodies in Guillain-Barré Syndrome*, 9 J. Clin. Lab. Anal. 308 (1995), filed on May 9, 2024 as Ex. 29-6 (ECF

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<sup>8</sup> “Hapten” is defined as “a small molecule, not antigenic by itself, that can react with antibodies of appropriate specificity and elicit the formation of such antibodies when conjugated to a larger antigenic molecule, usually a protein, called in this context the carrier or schlepper. Antibody production involves activation of B lymphocytes by the hapten and helper T lymphocytes by the carrier.” *Dorland’s Medical Dictionary Online*, <https://www.dorlandsonline.com/dorland/definition?id=21432&searchterm=hapten> (last accessed July 12, 2024).

No. 90-6) (“Terryberry”).<sup>9</sup> This is a foundational element of Petitioner’s theory, necessary to establish if the vaccine is to be found causal of disease (especially so, given the absence of evidence that the bacterial infection the vaccine seeks to protect against—*S. Pneumoniae*—is not associated with GBS). Whitton First Rep. at 18–20. As a result, such articles could well support causation in ways Dr. Steinman’s theory fell short in *Gamboa-Avila* and *Trollinger*.

But they do not. Al-Temeemi, for example, is not particularly recent (raising the question of why it was not previously offered—in this case or any of the other multiple parallel cases for that matter). In it, a group of Iraqi researchers performed a case-control study involving a particularly small sample size—eleven patients—to evaluate the presence of antiphospholipid antibodies in the blood serum of GBS patients. Al-Temeemi at 191. Their focus on this group of relevant antibodies stemmed from their determination that GBS was often comorbid in patients also suffering from other known autoimmune diseases, like lupus, and therefore “[n]early any neurological manifestation may occur in patients” who possess this particular antibody. *Id.* at 192. Al-Temeemi’s authors wished to evaluate the titer levels of the antiphospholipid antibodies in the subjects when compared to comparable healthy individuals, and at different times. *Id.*

Determinations about levels in comparison to timing amongst the sick subjects mostly produced statistically-insignificant results, although Al-Temeemi’s authors did observe a negative correlation between more recently-produced antibody levels and illness duration (consistent with articles discussed in prior comparable cases).<sup>10</sup> But even though Al-Temeemi noted (and at a statistically-significant level) higher antiphospholipid antibody levels in GBS patients in the initial illness stage than healthy controls, they deemed this likely attributable to “a more extensive immune reaction beyond the well known antiganglioside production, which has been related to the demyelination of the peripheral nerves.” Al-Temeemi at 198 (emphasis added). In other words, *Al-Temeemi did not find the phospholipid-targeting antibodies were pathogenic* in GBS. Indeed—Al-Temeemi speculates the antiphospholipid antibodies may be protective (based on different levels when evaluating severity of the GBS and the degree of treatment required in the studied patients). *Id.* Thus, Al-Temeemi (and based on extremely limited data) only supports *the existence* of this antibody in GBS patients, reaching no reliable conclusions about how it comes into existence—or whether it plays *any* pathogenic role.

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<sup>9</sup> Petitioner also filed an article referencing the presence of lipid-related antibodies in a small study set of GBS patients. See S. Mata et al., *Anti-GMI, Anti-Central Myelin Proteins, and Anti-Cardiolipin Autoantibodies During Plasma-Exchange in Guillain-Barré Syndrome (GBS)*, 13 J. Clin. Apheresis 155 (1998), filed as Ex. 29-5 (ECF No. 90-5) (“Mata”). But Mata deals with measurement of a different kind of phospholipid antibody – cardiolipin—and deems the antibody’s pathogenic role “less known” in comparison to anti-ganglioside antibodies. Mata at 155. Petitioner also has filed only a two-page excerpt from Mata, making it impossible to evaluate its findings completely.

<sup>10</sup> See *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2023 WL 35509, at \*36 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (stating that the articles “actually suggest that the phospholipid antigenic targets on the myelin either serve a protective, immune-modulating role, or at worst are damaged by an ongoing autoimmune process only after it has begun—thus greatly undermining the contention that antibodies to these lipid myelin components initiate it”).

Terryberry (yet another fairly old item of literature) evaluated the blood sera of a larger cohort (56 GBS patients), compared to controls that were healthy or suffered from other kinds of conditions also involving demyelination, with a focus on levels of 18 antibodies believed to be cross-reactive with myelin, including cardiolipin (a phospholipid antibody). Terryberry at 308–09. Nearly half of the studied subjects possessed anti-cardiolipin antibodies. *Id.* at 311. Moreover, many of the blood specimens tested positive for resolved *S. pneumoniae* infections—although this cannot be extrapolated to constitute proof of a causal association, since there would be no way to know when these prior infections occurred in association with GBS’s onset. Indeed, Terryberry’s authors observed that “some patients had antibodies to as many as 15 infectious agents.” *Id.* at 310, 312 (Tables 3 and 4). The study concluded, among other things, that its results “illustrate the difficulties in determining whether infection by a specific organism triggers the disease in individual patients”—a difficulty that has not diminished in the more than 25 years since Terryberry was published. *Id.* at 315.

Another “new” article was offered to bulwark Dr. Steinman’s contentions about the potentiality of an autoimmune cross-reaction between the vaccine’s conjugate and Contactin, which Dr. Steinman maintains “is targeted in some cases of GBS.” Second Steinman Rep. at 12; J. Devaux et al., *Nodal Proteins are Target Antigens in Guillain-Barré Syndrome*, 17 J. Periph. Nerv. Syst. 62 (2012), filed on May 9, 2024 as Ex. 29-7 (ECF No. 90-7) (“Devaux”). Devaux sought to obtain more information about to the degree to which the “nodes of Ranvier”—gaps along the otherwise-myelinated nerve axon—are targets of autoimmune attack in GBS, given that more is known about ganglioside structure-directed autoantibody attacks in driving the disease. Devaux at 62–63. It thus looked for evidence of autoantibodies against *different* proteins specific to these nodes, such as neurofascin (which is comparable to Contactin). *Id.* at 63. Blood sera from 150 Japanese patients (50 of whom were suffering from chronic immune demyelinating polyneuropathy, or “CIDP”—a distinguishable GBS variant) were tested on animal subjects, with findings that (a) the nodes were in fact targets, and (b) neurofascin was a specific target, but (c) anti-neurofascin antibodies were better associated with an axonal form of GBS (in which other antibodies to gangliosides were detected, and more likely were driving the disease in a primary manner), and (d) it could not be determined from the study “[t]he causes generating these autoantibodies in GBS,” with no identified antecedent illnesses. *Id.* at 67–70.

Thus, Devaux (like many other studies offered in the effort to prove the pneumococcal vaccine can cause GBS) sheds a dim light on another possible GBS-associated antibody in addition to anti-ganglioside antibodies. But it gives no additional reason to find the pneumococcal vaccine is likely *causal* of that antibody—let alone capable of producing it at the pathogenic levels necessary to drive disease, and in the absence of an existing/ongoing disease process as well. And the study’s focus was not on Contactin either, even if in a subset of patients’ antibodies to it were observed. Second Steinman Rep. at 13. Once again, Dr. Steinman offers an item of literature that

only glancingly supports his theory, observing a *plausible* connection but not appreciably supporting the conclusion that the identified GBS target is a likely disease driver.

Dr. Steinman's efforts to elevate the argument that the pneumococcal vaccine conjugate element might instigate production of autoimmune antibodies extended to other items of literature not previously filed. For example, he referenced two articles to support the argument that Caspr2<sup>11</sup> is “an antigen targeted in GBS.” Second Steinman Rep. at 23; R. Rosch et al., *Guillain-Barré Syndrome Associated with CASPR2 Antibodies: Two Paediatric Cases*, 19 J. Periph. Nerv. Syst. 246 (2014), filed on May 9, 2024 as Ex. 29-10 (ECF No. 90-10) (“Rosch”); X. Tan et al., *Guillain-Barré Like Syndrome: An Uncommon Feature of CASPR2 and LGII Autoimmunity*, 269 J. Neurol. 5893 (2022), filed on May 9, 2024 as Ex. 29-11 (ECF No. 90-11) (“Tan”).

Once again, these items are far less helpful than supposed. Rosch is a case report discussing two children (a two and six-year old) who developed GBS, both after different viral infections. Rosch at 246–47. The children in each case tested positive for autoantibodies against a Contactin-associated protein (which Dr. Steinman alleges could be the self mimic for the pneumococcal vaccine’s conjugate), but not the more-expected ganglioside antibodies. *Id.* at 247. However (and besides the fact that the subjects were children), neither had been vaccinated pre-onset. Rosch’s authors ultimately noted that although the reports supported the theory that these specific autoantibodies “*might potentially* contribute to peripheral nerve pathology in GBS,” existing studies involving these kinds of autoantibodies “reported to date have not characteristically involved demyelination.” *Id.* at 248. Thus, Rosch hardly stands for solid evidence of pathology—and certainly does not support an association between the pneumococcal vaccine and the production of anti-Contactin/Caspr2 antibodies.

Tan is a case report involving an adult who tested positive for the anti-Contactin antibodies proposed by Dr. Steinman as potentially causal herein, and who experienced a “GBS-like” syndrome (although the patient’s presentation was more typical of a different neurologic condition, Morvan syndrome<sup>12</sup>). Tan at 5893. Indeed, Tan noted the relevant autoantibodies were *more* associated with Morvan syndrome. *Id.* at 5894. Tan otherwise listed a few other comparable case reports (including Rosch). *Id.* at 5896–97. As with Rosch, Tan stressed the possibility that the identified autoantibodies might play some causal role in GBS, but observed the need for further

<sup>11</sup> CASPR2 is a “neuronal cell surface protein.” CASPR2 antibodies are “usually associated with peripheral nervous system involvement.” Rosch at 246.

<sup>12</sup> Morvan syndrome is a manifestation of syringomelia, “a slowly progressive syndrome of cavitation in the central segments of the spinal cord, generally in the cervical region, but sometimes extending up into the medulla oblongata” that “results in neurologic deficits, usually segmental muscular weakness and atrophy with a dissociated sensory loss.” In Morvan Syndrome, “the subcutaneous tissues of the hands become thickened, edematous, soft, swollen, cyanotic” along with “analgesic ulceration of the fingertips and paresthesia and atrophy of the hands and forearms.” *Dorland’s Medical Dictionary Online*, <https://www.dorlandsonline.com/dorland/definition?id=48639> and <https://www.dorlandsonline.com/dorland/definition?id=111022> (last accessed July 12, 2024).

study. *Id.* at 5899. Tan otherwise makes no mention of the propensity for *any* vaccine to trigger production of anti-Contactin antibodies in levels sufficient to become pathologic. Thus, these two articles are weak evidence that the identified antibodies more likely than not “can cause” GBS.

Only one of the not-previously-filed articles was actually published too recently to have been offered in prior cases like *Gamboa-Avila*. See D. Erkan et al., *Clinical Manifestations of Antiphospholipid Syndrome*, www.uptodate.com (Apr. 2024), filed on May 9, 2024 as Ex. 29-14 (ECF No. 90-14) (“Erkan”). Dr. Steinman referenced Erkan to justify his reliance on MS or other central nervous system-specific diseases and the antibodies thought causal in those contexts. Second Steinman Rep. at 28. But Erkan is an overview/review article discussing a distinguishable autoimmune condition, “antiphospholipid syndrome,” which it defines as a “multisystem disorder characterized by arterial, venous, or small vessel thromboembolic events, pregnancy morbidity, and/or nonthrombotic events (e.g., thrombocytopenia) in the presence of persistent antiphospholipid antibodies.” Erkan at 1. Thus, Erkan at the outset involves an illness *wholly unrelated* to GBS, and it pertains to this case solely because anti-phospholipid antibodies are theorized there as pathogenic *in a distinguishable context*. It bears not at all on the possibility that this antibody could promote an inflammatory neurologic condition like GBS; indeed, Erkan admits that those neurologic disorders suspected to be associated with the antibody (stroke, transient ischemic attack, epilepsy, chorea, migraines, etc.) have never been persuasively so linked. *Id.* at 5, 11.

#### B. Respondent’s Experts

Respondent offered two written reports,<sup>13</sup> including a single report authored by Lindsay Whitton, Ph.D.—an immunologist and Dr. Steinman’s counterpart in the *Trollinger* and *Gamboa-Avila* cases. Report, dated November 30, 2022, filed as Ex. A (ECF No. 73-1) (“Whitton Rep.”). Like Dr. Steinman’s showing, however, Dr. Whitton’s report filed in this case is almost wholly consistent with what he offered in these prior matters. *Gamboa-Avila*, 2023 WL 6536207, at \*10-15; *Trollinger*, 2023 WL 2521912, at \*13-18. I therefore do not include an extensive review or summary of the opinions he has offered in this case.

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<sup>13</sup> The second of Respondent’s reports was prepared by Dr. Thomas Leist. Report, dated November 8, 2022, filed as Ex. C (ECF No. 74-1). However, Dr. Leist is primarily a neurologist, and while he possesses sufficient general expertise to comment on proposed immunologic causes of GBS, I find his opinion to have overlapped Dr. Whitton’s to a large degree, but without adding any details worthy of evaluation (and without Dr. Whitton’s demonstrated specific competence in addressing questions of biochemistry and immunology). Accordingly (and also since I have now repeatedly found the causal theory offered herein deficient solely on the basis of the arguments Dr. Whitton raises), I do not include a discussion of Dr. Leist’s opinion.

### III. Procedural History

As noted, the Petition was filed approximately four years ago. Pet. at 1. Once most documents deemed necessary to adjudicate the claim had been filed, the matter was activated out of “pre-assignment review” in the spring of 2021 and initially assigned to the “special processing unit,” since it facially involved the kind of claim often easily settled (GBS as an injury after receipt of the flu vaccine). But after Respondent filed a Rule 4(c) Report in September 2021 contesting Petitioner’s right to compensation (ECF No. 34), the claim was transferred to my own docket, and I set a schedule for the filing of expert reports (while noting that the timeframe between the Petitioner’s receipt of the flu vaccine and GBS onset was too attenuated to support a Table claim, meaning the claim should be limited to causation associated with the pneumococcal vaccine). Order, dated December 7, 2021 (ECF No. 64). Expert reports were filed through the summer of 2023, and then I set a deadline for briefing the claim. The matter has been ripe for resolution since the filing of Petitioner’s reply in March of this year.

### IV. Parties’ Briefs

#### *Petitioner*

Petitioner maintains that he has met his causation-in-fact burden based on the factors established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005); Mot. at 48; Reply at 4.

Petitioner breaks down his causation theory into two parts: one involving the proposal that the pneumococcal vaccine sparks a cross-reaction as a result of antibodies produced in reaction to phosphoglycerol/phosphocholine found in at least one of the vaccine’s polysaccharide serotypes, and the second focusing on the conjugate. Mot. at 33, 43. With respect to the former, Petitioner relies on studies that he contends show increased levels of anti-phospholipid antibodies in the blood of GBS patients, though he posits that these studies do not determine the role of the antibodies in the pathogenesis of GBS. B. Gilburd et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with the Guillain-Barré Syndrome: Cross Reactivity of Pathogenic?*, 16 Autoimmunity 23 (1993), filed on October 6, 2022 as Ex. 28-11 (ECF No. 70-12) (“Gilburd”); G. Nakos et al., *Anti-phospholipid Antibodies in Serum From Patients with Guillain-Barré Syndrome*, 31 Intensive Care Med. 1401 (2005), filed on October 6, 2022 as Ex. 28-12 (ECF No. 70-13) (“Nakos”). He then notes that some of the vaccine’s serotypes have been shown not only to contain these phospholipid-oriented molecules, but that they are critical to immunogenicity. *Id.* at 35–37, citing J. Chang et al., *Relevance of O-acetyl Phospoglycerol Groups for the Antigenicity of Streptococcus Pneumoniae Serotype 18C Capsular Polysacchaaride*, 30 Vaccine 7090 (2012), filed on October 6, 2022 as Ex. 28-13 (ECF No. 70-14) (“Chang”). Finally, he maintains that antibodies are generated in response to the phosphoglycerol molecules in at least one of the vaccine’s 13 serotypes. *Id.* at 38–43. Thus, he contends that via the widely-accepted biologic mechanism of molecular mimicry, these

subcomponents of the vaccine trigger the production of autoantibodies specific to polar head structures in the myelin, thereby mistakenly attacking them, relying both on what is generally known about molecular mimicry in the context of GBS and other wild infections, like *C. jejuni*, and cases (discussed below) where this theory has been accepted. *Id.*

Petitioner spends comparatively less time discussing the aspect of his theory relying on the vaccine conjugate. He explains, however, its essence: that Dr. Steinman's database research had established two possible amino acid sequences that were shared by the conjugate and Contactin-1, a protein found in myelin, and that the lengths of these sequences were sufficient for a cross-reaction to occur. Mot. at 43–44. But this aspect of Dr. Steinman's opinion does not possess anywhere near the same degree of independent medical or scientific support suggesting any actual evidence of the theorized cross-reactivity, and Petitioner's brief does not cite any items of literature to this specific end.

In addition, Petitioner's brief asserts that certain items of literature actually reveal medical community support for a pneumococcal vaccine-GBS association—although the referenced articles make no such affirmative finding, and the readings given them by Petitioner are somewhat strained. Mot. at 47. One, for example, was deemed by Dr. Steinman as at least “reassuring” in its determination that the vaccine is not associated with a disproportionate number of GBS cases, but he maintained (especially since it relies on VAERS data) that it could not disprove an association. *Id.* P. Haber et. al., *Post-licensure Surveillance of 13-valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged P19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012–December 31, 2015*, 34 Vaccine 6330 (2016), filed on October 6, 2022 as Ex. 28-32 (ECF No. 70-33). And another did not involve the relevant vaccine, and had other limitations. *Id.* at 25.

#### *Respondent*

Respondent denies that the first *Althen* prong has been satisfied. Opp. at 14. The fact that the underlying *S. pneumoniae* bacterium is not associated with GBS is a fatal flaw to the theory, and nothing offered specific to it could overcome this foundational problem. *Id.* at 15–16. This might be because the kinds of molecules found in other bacteria that *are* so associated, like *C. jejuni*, express a different kind of molecule *known* to mimic aspects of myelin. *Id.* And Petitioner did not offer evidence associating the pneumococcal vaccine with GBS, while the existing epidemiology undercuts an association. *Id.* at 16–18.

The theory offered herein otherwise relies on unsubstantiated contentions about molecular mimicry that have been rejected in prior cases also involving the pneumococcal vaccine (as discussed below). Opp. at 20. In particular, it assumes without preponderant evidence that (a) phospholipid polar head groups can be a target of GBS much like ganglioside structures (even

though substantially more science supports the latter, with hardly any going the other way), and (b) antibodies to these polar head groups can drive disease, simply based on the fact that in a few studies they have been found in the blood of GBS patients. *Id.* at 21–22, citing Nakos, Gilburd. Respondent further criticizes Dr. Steinman’s reliance on the Bryson study, noting that it does not provide any connection between the immunogenicity of phosphoglycerol and the pathogenesis of GBS. *Id.* at 22–23; S. Bryson et al., *Structures of Preferred Human IgV Genes-Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation*, 196 J. Immunology 4723 (2016), filed on October 6, 2022 as Ex. 28-16 (ECF No. 70-17).

Respondent further criticized Dr. Steinman’s BLAST searches, which were specific to the conjugate “side” of his theory, as not probative of causation. Opp. at 24. Not only did Dr. Steinman not show that Contactin-1 is a likely GBS-instigating target, but he failed to acknowledge that sequence homology occurs “frequently between human tissues and antigens without pathological significance,” as explained by Dr. Whitton. *Id.* Further, he relied on case reports that were factually distinguishable, since they involved GBS variants believed to be driven by different antibodies and/or involving different antigenic targets. *Id.* at 23; Y. Miura et al., *Contactin-1 IgG4 Associates to Chronic Inflammatory Demyelinating Polyneuropathy with Sensory Ataxia*, 138 Brain 1484 (2015), filed on October 6, 2022 as Ex. 28-20 (ECF No. 70-21).

#### *Reply*

In his short reply, Petitioner reiterates the *Althen* prong one arguments made above. Reply at 3–4. He also attempts to refute Respondent’s remarks regarding Dr. Steinman’s bias towards Petitioners devaluing his theories, listing cases in which “theories posited by Petitioner’s expert withstood the appropriate scrutiny.” *Id.* at 2. He then responds to criticism of Dr. Steinman’s BLAST search methods, noting that is only one portion of a multi-step research process. *Id.*

## V. Applicable Law

### A. Standards for Vaccine Claims

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a “Table Injury”—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a “Non-Table Injury”). See Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); see also *Moberly v. Sec’y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).<sup>14</sup>

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<sup>14</sup> Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec’y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings

In this case, Petitioner cannot assert a Table claim (as there is no such claim with respect to the pneumococcal vaccine).

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*, 418 F.3d at 1278: “(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury.”

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a “reputable medical theory,” demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner’s theory must be based on a “sound and reliable medical or scientific explanation.” *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be “legally probable, not medically or scientifically certain.” *Knudsen*, 35 F.3d at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and

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concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at \*7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

thus scientific evidence offered to establish *Althen* prong one is viewed “not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has *consistently rejected* the contention that it can be satisfied merely by establishing the proposed causal theory’s scientific or medical *plausibility*. *See Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at \*2 (Fed. Cir. June 20, 2024) (arguments “for a less than preponderance standard” deemed “plainly inconsistent with our precedent” (citing *Moberly*, 592 F.3d at 1322)); *Boatman v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (“[h]owever, in the past we have made clear that simply identifying a ‘plausible’ theory of causation is insufficient for a petitioner to meet her burden of proof” (citing *Moberly*, 592 F.3d at 1322)); *see also Howard v. Sec'y of Health & Hum. Servs.*, 2023 WL 4117370, at \*4 (Fed. Cl. May 18, 2023) (“[t]he standard has been preponderance for nearly four decades”), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* “addresses the petitioner’s overall burden of proving causation-in-fact under the Vaccine Act” by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine “did cause” injury, the opinions and views of the injured party’s treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (“medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”)(quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“there is nothing . . . that mandates

that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at \*17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied*, (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

#### B. Analysis of Expert Testimony

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 594–96 (1993). See *Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). “The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.” *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. See e.g., *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner’s case. Where both sides offer expert testimony, a special master’s decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); see also *Isaac v. Sec'y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at \*17 (Fed. Cl. Spec. Mstr. July 30, 2012), mot. for rev. denied, 108 Fed. Cl. 743 (2013), aff’d, 540 F. Appx. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert’s credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); see also *Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

Expert opinions based on unsupported facts may be given relatively little weight. See *Dobrydnev v. Sec'y of Health & Hum. Servs.*, 556 F. Appx. 976, 992–93 (Fed. Cir. 2014) (“[a] doctor’s conclusion is only as good as the facts upon which it is based”) (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp.*, 509 U.S. 209, 242 (1993) (“[w]hen an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert’s opinion”)). Expert opinions that fail to address or are at odds with contemporaneous medical records may therefore be less persuasive than those which correspond to such records. See *Gerami v. Sec'y of Health & Hum. Servs.*, No. 12-442V, 2013 WL 5998109, at \*4 (Fed. Cl. Spec. Mstr. Oct. 11, 2013), aff’d, 127 Fed. Cl. 299 (2014).

C. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision”) (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. Appx. 875, 884 (Fed. Cir. 2013) (“[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered”).

D. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at \*21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at \*2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

## ANALYSIS

I have now ruled on *four* prior occasions that it is preponderantly *unlikely* that the pneumococcal vaccine “can cause” GBS. *See, e.g., Gamboa-Avila*, 2023 WL 6536207, at \*24; *Trollinger*, 2023 WL 2521912, at \*25; *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at \*34 (Fed. Cl. Spec. Mstr. Dec. 9, 2022); *Deshler v. Sec'y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at \*19 (Fed. Cl. Spec. Mstr. July 1, 2020). In each instance, I conducted a detailed review of the medical/scientific support offered for the proposed causation theory—and as noted above, in two of these prior cases Dr. Steinman offered an opinion

favoring causation (also opposed, as here, by Dr. Whitton). *Gamboa-Avila*, 2023 WL 6536207, at \*26–29; *Trollinger*, 2023 WL 2521912, at \*27–30.<sup>15</sup>

As already observed, the causation theory that was at issue in these earlier-decided matters is virtually the same as what has been proposed in this case. In *Bielak*<sup>16</sup> and *Trollinger*, petitioners argued that phosphoglycerol components of the pneumococcal vaccine caused antibodies to cross-react against relevant portions of the myelin, and both also involved nearly all of the same items of literature, like Ho, Gilburd, Nakos, etc. See *Trollinger*, 2023 WL 2521912, at \*19; *Bielak*, 2023 WL 35509, at \*15–17, 33–34; P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation*, 4 Sci. Translational Med. 1 (2012), filed on October 6, 2022 as Ex. 28-7 (ECF No. 70-7).

My determination in *Deshler*, 2020 WL 4593162, also bears on the outcome herein. The *Deshler* petitioner relied on the second component of Dr. Steinman’s theory: that the conjugate component of the Prevnar-13 vaccine *itself* had caused an aberrant, mimicry-driven autoimmune process relating to the vaccine’s antigens. *Deshler*, 2020 WL 4593162, at \*27. That petitioner’s expert opined that the subsequent B cell reaction (the primary goal of the vaccine) was driven by the polysaccharide component of the vaccination, although (unlike this case) he conceded that he could not demonstrate mimicry between the *S. pneumoniae* polysaccharides and self-structures. *Id.* Respondent’s expert in *Deshler* (Dr. Whitton again) argued in reaction that the polysaccharides contained in the vaccine did not share structural homology with self-structures of the peripheral nervous system, and thus could not contribute to the pathogenesis of GBS via a molecular mimicry-driven cross-reaction to the vaccine’s antigens. *Id.* I concurred with Respondent, while also finding that the petitioner relied too heavily on the temporal association between vaccination and onset as evidence of causation (and that there was another potential explanation for the claimant’s GBS that had not been rebutted). *Id.* at \*22, 27.

Most recently, I again denied entitlement in *Gamboa-Avila*. This time (and based on the wishes of the parties), I held an oral argument, to provide counsel the chance to articulate verbally how and why that claimant had met the relevant evidentiary standard. *Gamboa-Avila*, 2023 WL 6536207, at \*18. In turn, this permitted me the opportunity to address questions I had about the preponderant legal standard as applicable in a causation case—questions that went to the heart of the adequacy of the petitioner’s evidentiary showing. *Id.* I again found the causation theory

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<sup>15</sup> At the same time, Dr. Steinman has also been the expert in virtually *every* case decided by a different special master involving the same theory. See, e.g., *Sprenger v. Sec'y of Health & Hum. Servs.*, No. 18-279V, 2023 WL 8543435 (Fed. Cl. Spec. Mstr. Nov. 14, 2023); *Pierson v. Sec'y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Koller v. Sec'y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947 (Fed. Cl. Spec. Mstr. Oct. 8, 2021).

<sup>16</sup> Although the *Bielak* petitioner did not utilize Dr. Steinman, his expert borrowed the literature that Dr. Steinman offered in *Trollinger* and *Gamboa-Avila*. *Bielak*, 2022 WL 18058244, at \*8–9.

deficient, however. *Id.* at \*29. I also observed that the petitioner’s legal arguments leaned heavily on the concept that weighing the petitioner’s showing against Respondent’s, but finding the evidence did not preponderate in petitioner’s favor, was in effect a *heightening* of the legal standard—an argument that would, if accepted, seem to require special masters to find for petitioners in *any case* involving molecular mimicry and some showing of homology (in the absence of evidence that the literature offered to support the theory was suspect, or the homology showing otherwise in error). I rejected that contention as legally erroneous. *Id.* at 29–31.<sup>17</sup>

Ultimately (and as I have now explained several times in prior decisions), the theory that the pneumococcal vaccine “can cause” GBS seeks to demonstrate a link between indirectly-present components<sup>18</sup> of the vaccine and molecules found in myelin structures (in this case, lipids), but without additional sufficient evidence persuasively establishing likely pathogenicity, and with an over-reliance on either a showing of possible molecular homology or the presence of antibodies theorized to be produced by the vaccine (but certainly not intended to) in some limited studies involving GBS patients. In so arguing, claimants seek application of the flu vaccine-GBS association “template” in a context in which it does not work. To accept it would be to find causation mostly because a claimant’s GBS *post-dated* vaccination, reverse-engineering causation from the temporal association plus some molecular similarities, but without compelling evidence that a pathogenic reaction is likely.

Because of the foregoing, my skepticism about the viability of the causation theory presented *in this case* does not reflect personal bias, but is the product of my actual experience considering near-identical theories.<sup>19</sup> Admittedly, I am not bound to reach the same conclusion here despite my decisions in parallel matters. A new “twist” on the proposed theory, coupled with more recent studies or scientific findings, a different expert’s thinking on the topic, or even different facts about the claimant’s medical history, might justify reconsideration of a theory I have rejected many times before. But it is otherwise appropriate for me to rely on my learned experience as a special master adjudicating comparable cases, and to apply that experience in resolving comparable subsequent matters. *Doe v. Sec’y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338–39

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<sup>17</sup> I acknowledge that *Gamboa-Avila* is currently on appeal at the Federal Circuit, and there is thus a chance my reasoning will be rejected. However, having considered the theory so many times, and having had it challenged twice at the Court, it is fair to rely on my prior determinations as likely holding, and hence not to await a Circuit ruling before dismissal of this claim.

<sup>18</sup> By indirect, I mean only that the vaccine is not manufactured or engineered to *specifically include* phospholipids or phosphoglycerol—although the polysaccharide antigens in the vaccine *themselves* happen to contain these sub-structures, and their existence *within* the polysaccharides might have some internal significance.

<sup>19</sup> I am not treating my prior determinations akin to a system of precedential “claim preclusion,” whereby earlier determinations control the outcome in all similar cases to come. My earlier decisions do *not* have such legal preclusive force. But to take note of my familiarity with the relevant theory, and to require a claimant to establish how the theory before me differs in substance from what I have previously—and exhaustively—considered, is not the same a pre-deciding a subsequent case.

(2007) (“[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is *most probative of a claim*”) (emphasis added). It would be a dereliction of my duty to plug my ears, pretend such prior decisions were never issued, and “reinvent the wheel” in evaluating this claim. The Program does not demand that special masters start over, so to speak, each time a new case is filed, and ignore what they have already learned about an oft-repeated theory of vaccine injury causation.

Was anything offered in this case *different* than the evidence put forth in the prior matters? Recent Federal Circuit precedent suggests that even when taking into account experience resolving prior relevant cases, special masters should still ensure that their determinations are ultimately based on the evidence before them *in the relevant case*. See *Kalajdzic*, 2024 WL 3064398, at \*3.

As a result, I have carefully reviewed the new versions of Dr. Steinman’s reports filed herein, looking for any arguments that addressed the kinds of prior concerns I had voiced in *Gamboa-Avila* or *Trollinger* about the theory’s deficiencies. I also scrutinized items of literature not previously offered in the comparable earlier cases—and I included my review of many of those items in my otherwise-abbreviated summary of Dr. Steinman’s reports above.

Having performed this exercise, I do not find that the new versions of Dr. Steinman’s reports advance Petitioner’s contentions better than what I have considered before. The reports cite no recently-published scientific studies or reports more directly linking the pneumococcal vaccine to GBS, let alone the vaccine’s wild infectious counterpart. They also do not persuasively expand on Dr. Steinman’s contentions about phospholipid targets for GBS-driving antibodies created in reaction to the pneumococcal vaccine. And any items of literature not previously offered in cases I have decided do not demonstrably increase the likelihood Petitioner is correct.

For example, Al-Temeemi and Terryberry do not persuasively support the conclusion that GBS is likely driven by phospholipid-instigated, autoantibody attacks on antigenic nerve structure targets previously-unknown to the medical scientists who specifically study peripheral neuropathies, but which Dr. Steinman has somehow (and tellingly, solely in conjunction with his expert efforts in this case—not as a result of his own personal research in the field) uncovered. Others, like Rosch and Tan, are mere case reports unworthy of significant weight in determining causality as a general matter. *Pearson v. Sec’y of Health & Human Servs.*, No. 17-489V, 2019 WL 1150044, at \*11 (Fed. Cl. Spec. Mstr. Feb. 7, 2019) (concluding that case reports receive only limited evidentiary weight and cannot cure Althen prong one deficiencies); *see also Harris v. Sec’y of Health & Human Servs.*, No. 10-322V, 2014 WL 3159377, at \*18 (Fed. Cl. Spec. Mstr. June 10, 2014) (noting that “case reports are generally not a valuable form of evidence”)). And these reports at best only link anti-Contactin antibodies to GBS, while saying nothing about GBS occurring in the context of pneumococcal vaccine administration (let alone the wild bacterial

infection). I also (again) note that recent studies specific to MS and the Epstein-Barr Virus—which Dr. Steinman has in many prior cases cited to bulwark his general contentions about the pathologic character of molecular mimicry—are not otherwise relevant to a case involving a *different* vaccine and a *different* injury. Nothing recently published and offered in this case would render the proposed association between the pneumococcal vaccine and GBS more likely.

Thus, the expert reports filed in this case, along with their scientific and medical references, do not establish grounds for taking a *fifth look* at my prior, comprehensive determinations that *the pneumococcal vaccine cannot likely cause GBS*. It still has not been credibly established that anti-phospholipid or phosphoglycerol antibodies likely *drive* GBS—the lynchpin to a determination that the vaccine could spark it in the first place, by causing the pathogenic antibodies to come into existence. All Dr. Steinman has done is establish the existence of *some* biochemical similarities between molecular components of the vaccine’s antigens and nerve components. This cannot be enough to preponderantly show causation (and if it is, then all special masters may as well conclude that *all* covered vaccines can cause GBS equally, since a showing of molecular mimicry would never fail to establish homology, given how common it is in nature). And arguments about cross-reactivity between the conjugate in the vaccine and nerve components has been reasonably addressed, but rejected, in other prior cases like *Deshler*; at most, Dr. Steinman merely identifies a possible homologous mimic, but does not establish causation would likely flow from this showing. *Deshler*, 2020 WL 4593162, at \*20.

I also repeat what I have noted previously about my rejection of contrary determinations from other special masters who have found that the pneumococcal vaccine can cause GBS. *Gamboa-Avila*, 2023 WL 6536207, at \*25;<sup>20</sup> compare *Cooper v. Sec'y of Health & Hum. Servs.*, No. 18-1885V, 2024 WL 1522331 (Fed. Cl. Spec. Mstr. Mar. 12, 2024); *Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); *Maloney v. Sec'y*

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<sup>20</sup> One more recent such ruling underscored the overlap between my findings on the science and the contrary cases – perhaps with the intent of emphasizing how little actually separates my findings on this subject from my colleagues’ determinations. See *Cooper v. Sec'y of Health & Hum. Servs.*, No. 18-1885V, 2024 WL 1522331, at \*16 (Fed. Cl. Spec. Mstr. Mar. 12, 2024) (“even when reaching a different result, there has still been agreement that record evidence comparable to what has been presented in this case” establishes that “phyosphoglycerol is found in the pneumococcal vaccine; that the immune system produces antibodies in reaction to the relevant antigens containing the phosphoglycerol; and that individuals with neuropathies (although some suffer from the distinguishable disease MS) have been shown in small sample studies to possess antibodies specific to myelin-containing phospholipids” (quoting *Trollinger*, 2023 WL 2521912, at \*28)).

In fact, the varying outcomes in these cases more likely results from differences of opinion about proper application of the *Althen* prong one evidentiary standard. In effect, some special masters deem a merely plausible argument about mimicry – and one that does apply to the flu vaccine and GBS - enough to put “evidence on the scale” in a petitioner’s favor, ending the analysis. They seem to consider any counter-weighing of Respondent’s arguments (for why molecular mimicry cannot always persuasively explain every demyelinating post-vaccination injury, or why the impact of the pneumococcal vaccine cannot be deemed identical to the flu vaccine) to amount to an unfair heightening of the preponderant evidentiary standard. I, by contrast, view the evidence they call persuasive as inadequate – and its acceptance to be an exercise in *lowering* the preponderant standard.

*of Health & Hum. Servs.*, No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); *Pierson v. Sec'y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Koller v. Sec'y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947 (Fed. Cl. Spec. Mstr. Oct. 8, 2021).

I am of course not bound by these determinations—any more than the special masters who decided these claims were beholden to my analysis. But I otherwise do not find the legal reasoning expressed in those decisions to be persuasive. Rather, those decisions simply adopt the framework for the existing flu vaccine-GBS association, but without convincingly explaining why Respondent's counter-arguments merit less weight. I have also observed that these reasoned decisions cite the same items of literature that I have determined (after very close analysis) are not especially reliable or persuasive. *See, e.g., Bielak*, 2022 WL 18058244, at \*15–17, 32. Although future research or studies may better substantiate the alleged association between the pneumococcal vaccine and GBS, *existing* science on the subject is simply not favorable to the theory. To hold otherwise is to treat GBS as a “universal” vaccine injury, associated with virtually every covered vaccine, so long as the claimant has offered an expert opinion comparable to what Dr. Steinman has fashioned for this case.

## CONCLUSION

Claimants must carry their burden of proof. Here, Petitioner had the burden of preponderantly establishing that the pneumococcal vaccine can cause GBS. This has not been accomplished in this case. Accordingly, I deny entitlement.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.<sup>21</sup>

**IT IS SO ORDERED.**

/s/ Brian H. Corcoran  
Brian H. Corcoran  
Chief Special Master

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<sup>21</sup> Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.